Letters to the Editor

Clinical Characteristics of Ruptured Chordae Tendinae in Hospitalized Patients: Primary Tear Versus Infective Endocarditis

S. PORTUGESE, H. AMITAL, A. TENENBAUM, Y. BAR-DAYAN,
Y. LEVY, A. AFEK, J. SHEMESH, Y. SHOENFELD:
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To the Editor:

I read with interest the article on ruptured chordae tendinae by Portuguese *et al.* That mitral valve prolapse was present in 35% (7/20) of their patients was not unexpected. However, their finding of a very high incidence of 85% (6/7) with infective endocarditis was surprising.

I would like to offer two explanations for this unusual finding. First, this was a retrospective rather than a prospective study and thus suffers from the usual limitations and biases of a retrospective study. Second, this series consisted of hospitalized and therefore sicker patients only.

My personal experience is that the commonest cause of ruptured chordae tendinae is mitral valve prolapse¹ and that rupture of chordae tendinae may occur spontaneously in mitral valve prolapse without significant changes in symptomatology that prompt patients to be hospitalized.² Grenadier *et al.*³ reported that 75% of their patients with mitral valve prolapse and echocardiographic evidence of ruptured chordae tendinae did not have symptomatic deterioration. Very often I have seen spontaneous and often asymptomatic rupture of chordae tendinae in patients with mitral valve prolapse, either in the echocardiographic laboratory for evaluation of a murmur suspicious for mitral valve prolapse or in the operating room where the patients are undergoing mitral valve repair or replacement for significant mitral regurgitation.

Therefore, I would like to suggest that if Portuguese *et al.* would do a prospective study of all their patients with mitral valve prolapse, they would find more patients with spontaneous rupture of chordae tendinae than with infective endocarditis.

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Author's reply:

We thank Prof. Cheng for his comments. Our case series consisted of patients hospitalized because of severe symptoms due to the ruptured chordae tendinae. Thus, as Prof. Cheng mentioned, they were sicker than the patients in outpatient clinics and this may be the explanation for the high number of patients with mitral valve prolapse associated with infective endocarditis. Of interest is the study by Naggar *et al.*,¹ which followed 145 patients older than 60 years with mitral valve prolapse and demonstrated spontaneous rupture of chordae tendinae in 33 patients (23%). Four other patients had ruptured chordae tendinae associated with infective endocarditis.

We share Dr. Cheng's personal experience regarding unhospitalized patients with mitral valve prolapse. Indeed, the cases of ruptured chordae tendinae are by far more common than infective endocarditis.

More studies on this subject in hospitalized patients will clarify the issue.

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Pulmonary Embolism and Impending Paradoxical Embolism: A Role for Transesophageal Echocardiography?

C. ALEXIOU, R.A. HENDERSON, D. RICHENS: *Clin Cardiol* 1998;21:363–364

To the Editor:

Alexiou *et al.* describe a patient presenting subacute massive pulmonary embolism with impending paradoxical embolism across the interatrial septum. The patient did not receive thrombolytic therapy and died 14 h after an emergency embolectomy. The authors suggest that his poor preoperative hemody-

namic status led to a fatal outcome. Thus, any therapeutic attempt to relieve the right heart failure and circulatory shock could increase the survival rate after this kind of event.

Capellier *et al.*¹ described the effects of inhaled nitric oxide (NO) in four patients with severe pulmonary embolism. All patients received standard treatment for massive pulmonary embolism and 5–20 ppm inhaled NO. They attributed a 17–47% decrease in pulmonary artery pressure and a 8–52% increase in cardiac output to NO therapy and suggested that NO might be a therapeutic option in acute pulmonary embolism. Although controlled trials are needed to clarify this issue,² other reports have also documented beneficial effects of inhaled NO in the critical setting.^{3,4} Furthermore, experimental data support these clinical observations. For example, inhaled NO attenuated the increase in pulmonary vascular resistance and blunted the decrease of cardiac output after massive air embolism in dogs.⁵ In addition, NO therapy showed positive hemodynamic effects after acute pulmonary microspheres embolism.^{6,7} Besides the selective pulmonary vasodilatory effects, there is evidence of NO-induced inhibitory effects on platelet adhesion and aggregation,⁸ possibly diminishing additional clot formation when emboli reach the lungs. Even though the relevance of these findings remains to be elucidated, we believe that NO therapy could partially reverse circulatory collapse during pulmonary embolism. Any side effects of NO would be rapidly reversed by withholding its administration.

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Author's reply:

We appreciate the comments made by Tanus-Santos and Moreno Junior. In our report we describe a 41-year-old man with subacute massive pulmonary embolism in whom transesophageal echocardiography demonstrated a large thrombus straddling the interatrial septum. Thrombolysis was not considered appropriate because of the risk of paradoxical embolism, and the patient was referred for surgery.

Tanus-Santos and Moreno Junior suggest that adjunctive use of inhaled nitric oxide might have resulted in selective reduction of pulmonary vascular resistance and relief of right heart strain, potentially leading to clinical improvement. However, clinical evidence to support their suggestion is limited to a few case reports. Estagnasie *et al.*¹ reported a fall in pulmonary artery pressure in a 74-year-old female patient with pulmonary embolism and paradoxical cerebral embolism, but the patient died within 48 h. Similar effects of nitric oxide were reported by Capellier *et al.*² in four patients with pulmonary embolism but only two survived to hospital discharge. We doubt the use of the inhaled nitric oxide would have substantially improved the final outcome in our patient.

Tanus-Santos and Moreno Junior also suggest that nitric oxide may have a beneficial effect via inhibiting platelet aggregation, but whether this effect is clinically worthwhile in patients with venous thromboembolism is uncertain. Nevertheless, we agree with Tanus-Santos³ and Capellier *et al.*⁴ that the role of inhaled nitric oxide as an adjunct to conventional treatment with thrombolysis and anticoagulants in the management of patients with massive pulmonary embolism warrants further investigation.

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